WHAT IS CLAIMED IS:

1. Synthetic peptides comprising the regulatory virus protein R (Vpr) of the human immunodeficiency virus type 1(HIV-1).

2. The method of Claim 1, wherein said that peptides are characterized as follows

2.1. as a 96 amino acid long Vpr protein (sVpr$^{1-96}$)

2.2. as a 47 amino acid long N-terminal peptide (sVpr$^{1-47}$)

2.3. as a 49 amino acid long C-terminal peptide (sVpr$^{48-96}$), as well as

2.4. fragments of those peptides thereof, for example

2.4.1. overlapping, approximately 15 amino acid long peptides for epitope-characterization and isoelectric focusing

2.4.2. approximately 20 amino acid long peptides for structural and functional characterization of individual domains of Vpr, especially

2.4.2.1. the peptide sVpr$^{1-20}$, and

2.4.2.2. the peptide sVpr$^{21-40}$.

3. Peptides of claims 1 and 2, wherein said that peptides are characterized as follows

3.1. the 96 amino acid full lenght Vpr-Protein sVpr$^{1-96}$ comprises the sequence:

H - Met - Glu - Gln - Ala - Pro - Glu - Asp - Gln - Gly - Pro - Gln - Arg - Glu - Pro - Tyr - Asn - Glu - Trp - Thr - Leu - Glu - Leu - Leu - Glu - Glu - Leu - Lys - Ser - Glu - Ala - Val - Arg - His - Phe - Pro - Arg - Ile - Trp - Leu - His - Asn - Leu - Gly - Gln - His - Ile - Tyr - Glu - Thr - Tyr - Gly - Asp - Thr - Trp - Ala - Gly - Val - Glu - Ala - Ile - Ile - Arg - Ile - Leu - Gln - Gln - Leu - Leu - Phe - Ile - His - Phe - Arg - Ile - Gly - Cys - Arg - His - Ser - Arg - Ile - Gly - Val - Thr - Arg - Gln - Arg - Arg - Ala - Arg - Asn - Gly - Ala - Ser - Arg - Ser - OH

3.2. the 47 amino acid long N-terminal Peptid sVpr$^{1-47}$

H - Met - Glu - Gln - Ala - Pro - Glu - Asp - Gln - Gly - Pro - Gln - Arg - Glu - Pro - Tyr - Asn - Glu - Trp - Thr - Leu - Glu - Leu - Leu - Glu - Glu - Leu - Lys - Ser - Glu

- Ala - Val - Arg - His - Phe - Pro - Arg - Ile - Trp - Leu - His - Asn - Leu - Gly - Gln - His - Ile - Tyr - NH_2

3.3. the 49 amino acid long C-terminal peptide sVpr$^{48-96}$

Glu - Thr - Tyr - Gly - Asp - Thr - Trp - Ala - Gly - Val - Glu - Ala - Ile - Ile - Arg - Ile - Leu - Gln - Gln - Leu - Leu - Phe - Ile - His - Phe - Arg - Ile - Gly - Cys - Arg - His - Ser - Arg - Ile - Gly - Val - Thr - Arg - Gln - Arg - Arg - Ala - Arg - Asn - Gly - Ala - Ser - Arg - Ser-OH

3.4. fragments of those peptides thereof consisting of 15 amino acid long peptides

3.4.1. sVpr$^{11-25}$

Gln - Arg - Glu - Pro - Tyr - Asn - Glu - Trp - Thr - Leu - Glu - Leu - Leu - Glu - Glu

3.4.2. sVpr$^{41-55}$

Asn - Leu - Gly - Gln - His - Ile - Tyr - Glu - Thr - Tyr - Gly - Asp - Thr - Trp - Ala

3.4.3. sVpr$^{46-60}$

Ile - Tyr - Glu - Thr - Tyr - Gly - Asp - Thr - Trp - Ala - Gly - Val - Glu - Ala - Ile

3.4.4. sVpr$^{56-70}$

Gly- al-Glu-Ala-Ile-Ile-Arg-Ile-Leu-Gln-Gln-Leu-Leu-Phe-Ile

3.5. as the approximately 20 amino acid long peptides

3.5.1. the peptide sVpr$^{1-20}$ in form of

sVpr$^{1-20}$(Asn^{5,10,14})

H-Met - Glu - Gln - Ala - Asn - Glu - Asp - Gln - Gly - Asn - Gln - Arg - Glu - Asn - Tyr - Asn - Glu - Trp - Thr - Leu-NH_2, and

3.5.2. the peptide sVpr$^{21-40}$ in form of

sVpr$^{21-40}$(Asn^{35})

H-Glu - Leu - Leu - Glu - Glu - Leu - Lys - Ser - Glu - Ala - Val - Arg - His - Phe - Asn - Arg - Ile - Trp - Leu - His-NH_2

4. Method for the production of synthetic peptides derived from the regulatory virus proteins R (Vpr) of the human immunodeficiency virus type 1 (HIV-1) according to claims 1 to 3, wherein said that the synthesis of C-terminal Vpr-peptides is performed on a serine resin using a Perkin Elmer synthesizers and all N-terminal peptides are synthesized on a polystyrene polyoxyethylen resin. All peptides were synthesized using FMOC protection group strategy.

5. Method of claim 4, wherein said that at the end of the synthesis protection groups are cleaved off using a cleavage mixture consisting of 95% trifluoracetic acid, 3% triisopropylsilane, and depending on the peptide sequence 2 to 5 % ethandithiol, and the resin was separated.

6. Methods of claims 4 and 5, wherein said that the raw peptides were purified by HPLC chromatography on a column of silica gel using a linear gradient of TFA and water in acetonitrile.

7. Application of synthetic peptides derived from the regulatory virus proteins R (Vpr) of the human immunodeficiency virus type 1 (HIV-1) for therapeutic and diagnostic use.

8. Application of claim 7

8.1. in biological assays

8.1.1. for the development of serological test systems

8.1.2. for the development of Vpr antigen capture ELISA (enzyme linked immune sorbent assays)

8.2. for the generation of antibodies directed against HIV peptide sequences

8.3. for the generation of anti-viral reagents

8.4. for the development of test systems to screen potential Vpr inhibitors.

8.5. for the development of cell culture and animal models to investigate pathological mechanism of Vpr

8.6. for structural analyses of Vpr and its domains

8.7. for application in *in vitro* assembly of viruses and the development of vectors for application in gene therapy.

9. Applications according to claims 7 and 8, wherein said that those proteins are derived from *s*Vpr in which the N-terminal domain is mutated in either one, some, or all of the four proline residues.

10. Application according to claims 7 to 9 for the production of poly- and monoclonal antibodies or antisera specific for Vpr.

11. Application claims 7 to 10 for the production of epitope-different Vpr specific antibodies.

12. Application according to claims 7 to 11 in serological test systems.

13. Application according to claims 7 to 12 in a Vpr antigen (Ag) ELISA.

14. Application according to claims 7 to 13 as standard antigen for the calibration of Vpr-Ag-ELISA techniques.

15. Application according to claims 7 to 8 for the detection and for the estimation of Vpr concentration of viral Vpr in peripheral blood of HIV infected individuals.

16. Application of *s*Vpr proteins according to claims 7 and 8 for *in vitro* test systems for the characterization of Vpr Inhibitors.

17. Application according to claims 7 and 8 for complementation of the function of endogenous viral Vpr in cell cultures infected with *vpr* deficient HIV mutants.

18. Application according to claims 7, 8 and 17 for complementation of the function of endogenous viral Vpr in cultures of primary human lymphocytes infected with *vpr* deficient HIV mutants.

19. Application according to claims 7, 8, 17 18 zur for complementation of the function of endogenous viral Vpr in cultures of primary human monocytes / macrophages infected with *vpr* deficient HIV mutants.

~~20. Application according to claims 7 to 19 for the characterization of reagents which~~
a) block the interaction of Vpr with cellular factors, like the glucocorticoid-receptor, transcription factors and other DNA interacting enzymes and factors;
b) block the transcription activating activity of Vpr, and regulate, interfere with or block the activity of Vpr on steroid hormones;
c) regulate, interfere with , or block the transport of Vpr on its own or in complex with other components of the HIV preintegration complex as well as regulate, interfere with, or block the encapsidation of Vpr in budding virus particles during the HIV assembly;
d) regulate, interfere with, or block the Vpr induced cell cycle arrest as well as regulate, interfere with, or block the effect of Vpr on cell differentiation and cell growth;

e) regulate, interfere with, or block the cytotoxic effect of Vpr;

f) regulate, interfere with, or block the ion channel activity of.

21. Application of *s*Vpr proteins according to claims 7 and 8 for *in vivo* test systems that characterize Vpr inhibitors.

22. Application of *s*Vpr proteins according to claims 7 and 8 in animal model studies for the characterization of functions according to claim 20.

23. Application of *s*Vpr proteins according to claims 7 and 8 for the production of concentrated peptide solutions.

24. Application of *s*Vpr proteins according to claims 7, 8 and 23 for the production of Vpr specific inhibitors.

25. Application of *s*Vpr proteins according to claims 7, 8, 21 and 24 for the application of structure stabilizing factors that reduce the flexibility of the N-terminal domain in Vpr.

26. Application according to claim 25, wherein said that those structure stabilizing factors are

a) the UBA2 domain of the DNA repairing enzyme HHR23A which bind to Vpr,

b) Fab fragments of Vpr specific immune globulins,

c) viral factors, especially components of the HIV-1 Gag polyprotein precursor $Pr55^{Gag}$ which interacts with Vpr during the process of Virus assembly; or

d) the human glycocorticoid receptor or components thereof.

27. Application of *s*Vpr proteins according to claim 7 for *in vitro* assembly of retroviral preintegration complexes.

28. Application of *s*Vpr proteins according to claims 7, 8 and 27 in *in vitro* or *in vivo* application of gene transfer methods.

29. Application of *s*Vpr proteins according to claims 7, 8 and 28 for transfection, integration into chromosomal and episomale host DNA or any other gene transfer methods in eukaryotic cells.

30. Application of *s*Vpr proteins according to claims 7, 8 and 28 for gene transfer using *in vitro* synthesized and / or manipulated genes and fragments thereof for the transfer into cells, tissues, and organisms, and any application for the purpose of gene therapy.